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File 305:Analytical Abstracts 1980-2002/Feb W4
(c) 2002 Royal Soc Chemistry
*File 305: Frequency of updates and Alerts changing to weekly.
See HELP NEWS 305.
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(c) 2002 Reed Business Information Ltd.
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RANK charge added; see HELP RATES 399.
  File 434:SciSearch(R) Cited Ref Sci  1974-1989/Dec
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      Set  Items  Description
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? s (age or aging or longevity)

Processing
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         5254497  AGE
          549647  AGING
           70525  LONGEVITY
      S1 5634036  (AGE OR AGING OR LONGEVITY)
? s s1 and (marker or loci or locus or mutation or polymorphism or microsatellite)

Processing
Processed  10 of  25 files ...
Completed processing all files
         5634036  S1
          675793  MARKER
          263318  LOCI
          466979  LOCUS
         1075517  MUTATION
          488311  POLYMORPHISM
           74882  MICROSATELLITE
       S2  177507  S1 AND (MARKER OR LOCI OR LOCUS OR MUTATION OR
                   POLYMORPHISM OR MICROSATELLITE)
? s s2 and ((chromosome(w) 4) or D4S1564 or d4s411 or d4s1572 or d4s2986 or d4s406
or d4s1611 or d4s414 or afm248zg9 or 248zg9 or z23817)

Processed  10 of  25 files ...
Processing
Completed processing all files
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         1058168  CHROMOSOME
        11784648  4
           16695  CHROMOSOME(W)4
              11  D4S1564
               1  D4S411
               0  D4S1572
               7  D4S2986
               2  D4S406
               7  D4S1611
              22  D4S414
               0  AFM248ZG9
               0  248ZG9
               0  Z23817
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                   OR D4S2986 OR D4S406 OR D4S1611 OR D4S414 OR AFM248ZG9 OR
                   248ZG9 OR Z23817)
 ? rd s3

 ...examined 50 records  (50)
 ...examined 50 records  (100)
 ...examined 50 records  (150)
 ...examined 50 records  (200)
 ...examined 50 records  (250)
 ...examined 50 records  (300)
 ...examined 50 records  (350)
 ...examined 50 records  (400)
 ...examined 50 records  (450)
 ...completed examining records
```

S4 265 RD S3 (unique items)

?

13337612 BIOSIS NO.: 200100544761
Genome-wide scan for **age** at onset of Alzheimer disease.
AUTHOR: Li Y(a); Saunders A M(a); Roses A D(a); Small G W; Scott W K(a); Conneally P M; Vance J M(a); Gilbert J R(a); Haines J L; Pericak-Vance M A(a)
AUTHOR ADDRESS: (a)Ctr Human Genetics, Duke Univ Medical Ctr, Durham, NC** USA
JOURNAL: American Journal of Human Genetics 69 (4 Supplement):p200 October, 2001
MEDIUM: print
CONFERENCE/MEETING: 51st Annual Meeting of the American Society of Human Genetics San Diego, California, USA October 12-16, 2001
ISSN: 0002-9297
RECORD TYPE: Citation
LANGUAGE: English
SUMMARY LANGUAGE: English

4/7/10 (Item 10 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13336898 BIOSIS NO.: 200100544047
Genome scan for blood pressure in Dutch dyslipidemic families reveals linkage to a **locus** on chromosome 4p.
AUTHOR: Allayee Hooman; de Bruin Tjerk W A; Dominguez K Michelle; Cheng Li S-C; Ipp Eli; Cantor Rita M; Krass Kelly L; Keulen Eric T P; Aouizerat Bradley E; Lusis Aldons J; Rotter Jerome I(a)
AUTHOR ADDRESS: (a)Division of Medical Genetics, Cedars-Sinai Medical Center, 8700 W Beverly Blvd, Los Angeles, CA, 90048: jrotter@xchg.peds.csmc.edu**USA
JOURNAL: Hypertension (Baltimore) 38 (4):p773-778 October, 2001
MEDIUM: print
ISSN: 0194-911X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: Genes contributing to common forms of hypertension are largely unknown. A number of studies in humans and in animal models have revealed associations between insulin resistance, dyslipidemia, and elevated hypertension. To identify genes contributing to blood pressure (BP) variation associated with insulin-resistant dyslipidemia, we conducted a genome-wide scan for BP in a set of 18 Dutch families exhibiting the common lipid disorder familial combined hyperlipidemia. Our results reveal a **locus** on **chromosome 4** that exhibits a significant lod score of 3.9 with systolic BP. In addition, this **locus** also appears to influence plasma free fatty acid levels (lod=2.4). After adjustment for **age** and gender, the lod score for systolic BP increased to 4.6, whereas the lod score for free fatty acid levels did not change. The **chromosome 4 locus** contains an attractive candidate gene, alpha-adducin, which has been associated with altered BP in animal studies and in some human populations. However, we found no evidence for an association between 2 intragenic alpha-adducin polymorphisms and systolic BP in this sample. We also observed suggestive evidence for linkage (lod=1.8) of diastolic BP to the lipoprotein lipase gene **locus** on chromosome 8p, supporting a finding previously observed in a separate insulin-resistant population. In addition, we also obtained suggestive evidence for linkage of systolic BP (lod=2.4) and plasma apolipoprotein B levels (lod=2.0) to a **locus** on proximal chromosome 19p. In conclusion, our genome scan results support the existence of multiple genetic factors that can

influence both BP and plasma lipid parameters.

4/7/12 (Item 12 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2002 BIOSIS. All rts. reserv.

13279636 BIOSIS NO.: 200100486785
Genome-wide linkage disequilibrium mapping of late onset Alzheimer's disease in Finland.
AUTHOR: Hiltunen M J(a); Mannermaa A; Thompson D; Easton D; Pirskanen M(a); Helisalmi S(a); Koivisto A M(a); Lehtovirta M(a); Ryynanen M; Soininen H (a)
AUTHOR ADDRESS: (a)Department of Neurology, Kuopio University and University Hospital, Kuopio**Finland
JOURNAL: Society for Neuroscience Abstracts 27 (1):p331 2001
MEDIUM: print
CONFERENCE/MEETING: 31st Annual Meeting of the Society for Neuroscience San Diego, California, USA November 10-15, 2001
ISSN: 0190-5295
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: In order to find novel susceptibility genes for late onset Alzheimer's disease (AD), we have performed a population based genome-wide search using linkage disequilibrium (LD) mapping. To avoid population stratification, 47 late onset AD patients and 51 **age** -matched controls were carefully chosen from the same geographical area in Eastern Finland. Initial genome-wide screening with 366 polymorphic **microsatellite** markers revealed 22 chromosomal **loci** associated with AD with P-values in the range of 0.05>P>0.001. Subsequent comparison of single allele frequencies of the **microsatellite** markers in the AD and control groups indicated the presence of risk alleles displaying suggestive association with AD (odds ratios >1). In addition, certain markers revealed significantly lower frequencies of particular alleles in the AD group than in the control group suggesting a protective effect conferred by these alleles against the development of AD (odds ratios <1). Screening of the 22 LD regions with additional **microsatellite** markers revealed eight chromosomal **loci** in 1p36.12, 2p22.2, 3q28, 4p13, 10p13, 13q12, 18q12.1 and 19p13.3 to be associated with AD with in more than one **microsatellite** **marker**. These data suggest that there exist several AD-associated chromosomal **loci**, which may encompass novel susceptibility genes for late onset AD.

4/7/13 (Item 13 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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13257537 BIOSIS NO.: 200100464686
A genome-wide scan for linkage to human exceptional **longevity** identifies a **locus** on **chromosome 4**.
AUTHOR: Puca Annibale A(a); Daly Mark J; Brewster Stephanie J; Matise Tara C; Barrett Jeffrey; Shea-Drinkwater Maureen; Kang Sammy; Joyce Erin; Nicoli Julie; Benson Erica; Kunkel Louis M; Perls Thomas
AUTHOR ADDRESS: (a)Genetics Division, Howard Hughes Medical Institute, Children's Hospital and Harvard Medical School, 300 Longwood Avenue, Boston, MA, 02115: apuca@rascal.med.harvard.edu**USA
JOURNAL: Proceedings of the National Academy of Sciences of the United States of America 98 (18):p10505-10508 August 28, 2001
MEDIUM: print
ISSN: 0027-8424

DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: Substantial evidence supports the familial aggregation of exceptional **longevity**. The existence of rare families demonstrating clustering for this phenotype suggests that a genetic etiology may be an important component. Previous attempts at localizing **loci** predisposing for exceptional **longevity** have been limited to association studies of candidate gene polymorphisms. In this study, a genome-wide scan for such predisposing **loci** was conducted by using 308 individuals belonging to 137 sibships demonstrating exceptional **longevity**. By using nonparametric analysis, significant evidence for linkage was noted for **chromosome 4** at **D4S1564** with a MLS of 3.65 (P=0.044). The analysis was corroborated by a parametric analysis (P=0.052). These linkage results indicate the likelihood that there exists a gene, or genes, that exerts a substantial influence on the ability to achieve exceptional old **age**. Identification of the genes in humans that allow certain individuals to live to extreme old **age** should lead to insights on cellular pathways that are important to the **aging** process.

4/7/17 (Item 17 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13135860 BIOSIS NO.: 200100343009
Genome-wide linkage analysis reveals evidence of multiple regions that influence variation in plasma lipid and apolipoprotein levels associated with risk of coronary heart disease.
AUTHOR: Klos Kathy L; Kardia Sharon L R; Ferrell Robert E; Turner Stephen T ; Boerwinkle Eric; Sing Charles F(a)
AUTHOR ADDRESS: (a)Department of Human Genetics, University of Michigan, 1241 E. Catherine St, 5928 Buhl Building, Ann Arbor, MI, 48109-0618: csing@umich.edu**USA
JOURNAL: Arteriosclerosis Thrombosis and Vascular Biology 21 (6):p971-978 June, 2001
MEDIUM: print
ISSN: 1079-5642
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: Results of genome-wide linkage analyses to identify chromosomal regions that influence interindividual variation in plasma lipid and apolipoprotein levels in the Rochester, Minn, population are reported. Analyses were conducted for total cholesterol (total-C), triglycerides (TGs), high density lipoprotein cholesterol (HDL-C), apolipoprotein A-I, apolipoprotein A-II, apolipoprotein B, apolipoprotein C-II, apolipoprotein C-III, apolipoprotein E, the total-C/HDL-C ratio, and the TG/HDL-C ratio. Genotypes were measured for 373 genome-wide **marker loci** on 1484 individuals distributed among 232 multigeneration pedigrees sampled without regard to health status. LOD scores and estimates of additive genetic variance associated with map locations were obtained by using the variance-component method of linkage analysis. No evidence of linkage with genes influencing variation in **age** served as a negative control. Plasma apolipoprotein E levels and the apolipoprotein E gene served as a positive control (LOD score 4.20). Evidence (LOD score >2.00) was provided that was suggestive of a gene or genes on chromosomes 4 and 5 influencing variation in the apolipoprotein A-II level, on chromosome 12 influencing variation in the apolipoprotein

A-I level, and on chromosome 17 influencing variation of total-C/HDL-C. These analyses provide new information about genomic regions in humans that influence interindividual variation in plasma lipid and apolipoprotein levels and serve as a basis for further fine-mapping studies to identify new genes involved in lipid metabolism.

4/7/21 (Item 21 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13114450 BIOSIS NO.: 200100321599
A new **locus** for autosomal recessive RP (RP29) mapping to chromosome 4q32-q34 in a Pakistani family.
AUTHOR: Hameed Abdul; Khaliq Shagufta(a); Ismail Muhammad; Anwar Khalid; Mehdi S Qasim; Bessant David; Payne Annette M; Bhattacharya Shomi S
AUTHOR ADDRESS: (a)Biomedical and Genetic Engineering Division, Dr. A. Q. Khan Research Laboratories, 25 Mauve Area, Islamabad: sqmehdi@ish.comsats.net.pk**Pakistan
JOURNAL: IOVS 42 (7):p1436-1438 June, 2001
MEDIUM: print
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: Purpose. To map the disease **locus** in a six-generation, consanguineous Pakistani family with autosomal recessive retinitis pigmentosa (arRP). All affected individuals had pigmentary retinopathy associated with symptoms of night blindness and the loss of peripheral visual fields by the **age** of 20 years, loss of central vision between the ages of 25 and 30 years, and complete blindness between the ages of 40 and 50 years. Methods. Genomic DNA from family members was typed for alleles at known polymorphic genetic markers using polymerase chain reaction. Alleles were assigned to individuals, which allowed calculation of LOD scores using the programs Cyrillic (http://www.cyrillicsoftware.com) and MLINK (Cherwell Scientific Publishing Ltd., Oxford, UK). The genes for membrane glycoprotein (M6a) and chloride channel 3 (CLCN3) were analyzed by direct sequencing for mutations. Results. A new **locus** for arRP (RP29) has been mapped to chromosome 4q32-q34. A maximum two-point LOD score of 3.76 was obtained for the **marker** D4S415, with no recombination. Two recombination events in the pedigree positioned this **locus** to a region flanked by markers D4S621 and D4S2417. A putative region of homozygosity by descent was observed between the **loci** D4S3035 and D4S2417, giving a probable disease interval of 4.6 cM. **Mutation** screening of two candidate genes, M6a and CLCN3, revealed no disease-associated mutations. Conclusions. The results suggest that the arRP phenotype maps to a new **locus** and is due to a mutated gene within the 4q32-q34 chromosomal region.

4/7/38 (Item 38 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12590209 BIOSIS NO.: 200000343711
Association between the alpha-adducin gene and hypertension in the HyperGEN Study.
AUTHOR: Province Michael A(a); Arnett Donna K; Hunt Steven C; Leiendecker-Foster Cathie; Eckfeldt John H; Oberman Albert; Ellison R Curtis; Heiss Gerardo; Mockrin Stephen C; Williams Roger R
AUTHOR ADDRESS: (a)Division of Biostatistics, Washington University School of Medicine, 660 S. Euclid, Saint Louis, MO, 63110**USA

JOURNAL: American Journal of Hypertension 13 (6 Part 1):p710-718 June, 2000
MEDIUM: print
ISSN: 0895-7061
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: This report from the HyperGEN Study, one of four networks participating in the NHLBI-sponsored Family Blood Pressure Program, presents the results of an association study based on 822 white and 572 black subjects (cases and controls) participating in the HyperGEN Network from five geographically diverse field centers. All cases met the Joint National Committee on Detection and Treatment of High Blood Pressure (JNC VI) criteria for hypertension (Stage I or higher). Each subject was clinically examined for risk factors for hypertension as well as genotyped for the point **mutation** Gly460Trp at the alpha-adducin **locus** on chromosome 4p. In the white group, the prevalence of genotypes with one or more Trp alleles was 26% in normotensives, versus 33% in hypertensives randomly selected from the population, and 39% among the multiply affected hypertensive sibships. Overall, in whites, the Trp allele significantly increased the odds of hypertension (P = .0056), with an odds ratio (OR) of 1.73 (95% confidence interval (CI) = 1.17, 2.54). The alpha-adducin gene remained a significant independent predictor of hypertension in a multivariate logistic model even after correcting for other risk factors for hypertension, including gender, **age**, body mass index (BMI), smoking, LDL cholesterol, triglycerides, urine sodium (Na), and urine potassium (K), (OR = 1.55, 95% CI = 1.03, 2.34). Through the use of regression trees, several gene-by-environment interactions were implicated, suggesting that alpha-adducin appears to be a particularly important risk factor (OR = 4.2) for older (**age** > 60.5 years), less lean (BMI < 25.8 kg/m2) subjects with moderately high triglycerides (between 145.5 and 218.5 mg/dL). In the black group, the relationship was less clear. Overall, it was protective against hypertension. The prevalence of genotypes with one or more Trp alleles was 24% among normotensive versus 11% in hypertensive black subjects randomly selected from the population, and 13% among multiply affected hypertensive sibships, resulting in an OR of 0.48 (P = .0231; 95% CI = 0.25, 0.90). However, the Trp genotype was no longer a significant independent predictor of hypertension risk in the multivariate logistic model (OR = 0.79; 95% CI = 0.37, 1.67), suggesting that it may be operating through one or more of these other factors. Thus, we conclude that the alpha-adducin gene is a significant, independent risk factor for hypertension in whites, but not in blacks, and may play a particularly important role for subjects with certain constellations of other risk factors.

12485764 BIOSIS NO.: 200000239266
Genetic approaches to the study of replicative senescence.
AUTHOR: Ran Qitao; Pereira-Smith Olivia M(a)
AUTHOR ADDRESS: (a)Roy M. and Phyllis Gough Huffington Center on Aging, Baylor College of Medicine, Huffington Center on Aging, 1 Baylor Plaza, Room N803, Houston, TX, 77030**USA
JOURNAL: Experimental Gerontology 35 (1):p7-13 Feb., 2000
ISSN: 0531-5565
DOCUMENT TYPE: Literature Review
RECORD TYPE: Abstract
LANGUAGE: English

SUMMARY LANGUAGE: English

ABSTRACT: Genetic analyses of replicative senescence have revealed the dominance of the senescent phenotype since whole cell fusion of normal with immortal cells yields hybrids having limited division potential. We exploited the recessive nature of immortality by fusing different immortal human cell lines with each other and identified four complementation groups for indefinite division. This allowed for a focussed approach involving microcell mediated chromosome transfer that led to the implication of chromosomes 1, 4 and 7 as **loci** for cell senescence genes. More recently we have cloned the gene on **chromosome 4**, MORF 4. It is a member of a family of genes with motifs suggestive of transcriptional regulators. Characterization of this novel gene family should lend further insights into the phenomenon of replicative cell senescence.

4/7/52 (Item 52 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12037464 BIOSIS NO.: 199900317983
Genetics of Parkinson's disease.
AUTHOR: Mizuno Y(a); Hattori N(a); Mori H(a)
AUTHOR ADDRESS: (a)Department of Neurology, Juntendo University School of Medicine, 2-1-1 Hongo, Bunkyo, Tokyo, 113**Japan
JOURNAL: Biomedicine & Pharmacotherapy 53 (3):p109-116 April, 1999
ISSN: 0753-3322
DOCUMENT TYPE: Literature Review
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: Here we review familial Parkinson's disease from clinical, as well as molecular genetic aspects. To date, two genes responsible for familial Parkinson's disease have been identified: one is the alpha-synuclein gene located in the long arm of **chromosome 4**, and the other is the parkin gene located in the long arm of chromosome 6. The mode of inheritance of the former is autosomal dominant and clinical features consist of levodopa-responsive parkinsonism; the **age** of onset is younger than that of the sporadic cases (in their 40s), and the progression is faster (average disease duration approximately nine years). The latter form is transmitted as an autosomal recessive, and clinical features consist of early onset (in their 20s), levodopa-responsive parkinsonism, and a slow progression of the disease. In addition, the tau gene has been shown to be the disease gene for familial frontotemporal dementia and parkinsonism linked to chromosome 17. There are many other clinical phenotypes of familial Parkinson's disease among which three forms have been mapped to certain chromosome **loci**: one is in the short arm of chromosome 2, the two other forms are in the different **loci** of the short arm of **chromosome 4**. All of them are transmitted as autosomal dominant traits manifesting levodopa responsive parkinsonism. There still exists however, other clinical phenotypes of chromosome **loci** which are not known. Molecular cloning of these familial Parkinson's disease genes and the elucidation of the functions of the proteins encoded will certainly contribute greatly to the investigation of the etiology and pathogenesis of more common sporadic form of Parkinson's disease.

4/7/57 (Item 57 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11802454 BIOSIS NO.: 199900048563
Evidence of an increased risk of hearing loss in heterozygous carriers in a Wolfram syndrome family.
AUTHOR: Ohata Tomoaki; Koizumi Akio(a); Kayo Tsuyoshi; Shoji Yutaka; Watanabe Arata; Monoh Katsumi; Higashi Koichiro; Ito Seiki; Ogawa Osamu; Wada Yasuhiko; Takada Goro
AUTHOR ADDRESS: (a)Dep. Hyg., Akita Univ. Sch. Med., Hondo 1-1-1, Akita 010-8543**Japan
JOURNAL: Human Genetics 103 (4):p470-474 Oct., 1998
ISSN: 0340-6717
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Wolfram syndrome (MIM 222300) is characterized by juvenile-onset diabetes mellitus and optic atrophy. Previous linkage analyses in the United States and UK families have indicated that the gene for Wolfram syndrome (WFS) is localized on the short arm of **chromosome 4.** We herein confirm the linkage of the WFS **locus** to D4S3023 on 4p with a two-point LOD score of 3.42 in a large Japanese family with Wolfram syndrome. Multipoint linkage analysis revealed the maximum LOD score of 4.82 between D4S3023 and D4S394. We also evaluated putative health risks in carriers by multiple logistic analysis with independent variables, **age**, gender, and numbers of affected haplotypes and with dependent variables, such as hearing loss, diabetes mellitus, polyuria, incontinence, psychological illness, and visual acuity. The results showed that the putative disease haplotype increased a risk of hearing loss (odds ratio =35.68, 95% confidence interval =4.12-308.95) and diabetes mellitus (odds ratio =7.57, 95% confidence interval =2.03-28.23) independently. This is the first report of an increased health risk of illness in carriers, other than for psychiatric disease.

4/7/58 (Item 58 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11729982 BIOSIS NO.: 199800511713
Genetics of psoriasis.
AUTHOR: Henseler Tilo(a)
AUTHOR ADDRESS: (a)Dep. Dermatol., Univ. Kiel, Schittenhelmstrasse 7, D-24105 Kiel**Germany
JOURNAL: Archives of Dermatological Research 290 (9):p463-476 Sept., 1998
ISSN: 0340-3696
DOCUMENT TYPE: Literature Review
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Non-pustular psoriasis consists of two disease subtypes, type I and type II, which demonstrate distinct characteristics. Firstly the disease presents in different decades of life, in type I before the **age** of 40 years and later in type II. Secondly, contrasting frequencies of HLA alleles are found: type I patients express predominantly HLA-Cw6, -B57, and -DR7, whereas in type II patients HLA-Cw2 is overrepresented. Finally, familial inheritance is found in type I but not in type II psoriasis. The study of concomitant diseases in psoriasis contributes to deciphering the distinct patterns of the disease. Defence against invading microorganisms seems better developed in psoriatics than in controls. This evolutionary benefit may have caused the overall high incidence of psoriasis of 2%. Psoriasis is a multifactorial and heterogenetically inherited disease. The heterogeneity is evident by the diversity of genetically linked markers. The multifactorial component results from the observation of external trigger mechanisms, such as the Koebner phenomenon, stress and the intake of

certain drugs. Twin studies have shown that environmental factors contribute to the onset of the disease. In type I psoriasis, special extended haplotypes such as EH57.1 (HLA-Cw6-B57-DRB1*0701-DQA1*0201-DQB1*0303) and EH65.1 (HLA-Cw8-B65-DRB1*0102-DQB1*0501) have been found to be increased. The application of **microsatellite** techniques has identified distinct positions on several chromosomes at which putative psoriasis genes may be located. Disease susceptibility genes are thought to be present on chromosomes 4q, 6p, 16q, 17q and 20p. Moreover, on chromosome 1q, genes regulating epidermal differentiation have been identified. Linkage to this area has been proposed. Furthermore, psoriasis gene **loci** on chromosomes 2, 8 and 20 have been suggested.

?

07657278 Genuine Article#: 192RP Number of References: 35
Title: A new **locus** for autosomal dominant Stargardt-like disease maps to **chromosome 4**
Author(s): Kniazeva M (REPRINT) ; Chiang MF; Morgan B; Anduze AL; Zack DJ; Han M; Zhang K
Corporate Source: UNIV COLORADO,HOWARD HUGHES MED INST, DEPT MOL CELLULAR & DEV BIOL, CAMPUS BOX 347/BOULDER//CO/80309 (REPRINT); WILMER EYE INST,DEPT OPHTHALMOL/BALTIMORE//MD/; JOHNS HOPKINS UNIV,SCH MED, DEPT MOL BIOL & GENET/BALTIMORE//MD/21205; JOHNS HOPKINS UNIV,SCH MED, DEPT NEUROSCI/BALTIMORE//MD/21205; ISL MED CTR,/ST CROIX//VI/
Journal: AMERICAN JOURNAL OF HUMAN GENETICS, 1999, V64, N5 (MAY), P 1394-1399
ISSN: 0002-9297 Publication date: 19990500
Publisher: UNIV CHICAGO PRESS, 5720 SOUTH WOODLAWN AVE, CHICAGO, IL 60637-1603
Language: English Document Type: ARTICLE
Abstract: Stargardt disease (STGD) is the most common hereditary macular dystrophy and is characterized by decreased central vision, atrophy of the macula and underlying retinal-pigment epithelium, and frequent presence of prominent flecks in the posterior pole of the retina. STGD is most commonly inherited as an autosomal recessive trait, but many families have been described in which features of the disease are transmitted in an autosomal dominant manner. A recessive **locus** has been identified on chromosome 1p (STGD1), and dominant **loci** have been mapped to both chromosome 13q (STGD2) and chromosome 6q (STGD3). In this study we describe a kindred with an autosomal dominant Stargardt-like phenotype. A genomewide search demonstrated linkage to a **locus** on chromosome 4p, with a maximum LOD scare of 5.12 at a recombination fraction of .00, for **marker** D4S403. Analysis of extended haplotypes localized the disease gene to an similar to 12-cM interval between **loci** D4S1582 and D4S2397. Therefore, this kindred establishes a new dominant Stargardt-like **locus**, STGD4.

4/7/126 (Item 29 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

01079544 Genuine Article#: FU901 Number of References: 22
Title: GENETIC-ANALYSIS OF INDEFINITE DIVISION IN HUMAN-CELLS - EVIDENCE FOR A CELL SENESCENCE-RELATED GENE(S) ON HUMAN **CHROMOSOME-4**
Author(s): NING Y; WEBER JL; KILLARY AM; LEDBETTER DH; SMITH JR; PEREIRASMITH OM
Corporate Source: BAYLOR UNIV,INST MOLEC GENET,DEPT CELL BIOL,DEPT MED,DIV MOLEC VIROL/HOUSTON//TX/77030; UNIV TEXAS,MD ANDERSON CANC CTR,DIV LAB MED/HOUSTON//TX/77030; MARSHFIELD MED RES FDN/MARSHFIELD//WI/54449; BAYLOR UNIV,INST MOLEC GENET,DEPT CELL BIOL,DEPT MED,DIV MOLEC VIROL/HOUSTON//TX/77030
Journal: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, 1991, V88, N13, P5635-5639
Language: ENGLISH Document Type: ARTICLE
Abstract: Earlier studies had demonstrated that fusion of normal with immortal human cells yielded hybrids having limited division potential. This indicated that the phenotype of limited proliferation (cellular senescence) is dominant and that immortal cells result from recessive changes in normal growth-regulatory genes. In additional studies, we exploited the fact that the immortal phenotype is recessive and, by fusing various immortal human cell lines with each other, identified four complementation groups for indefinite division. Assignment of cell lines to specific groups allowed us to take a focused approach to identify the chromosomes and genes involved in growth regulation that have been modified in immortal cells. We report here that introduction of a normal human **chromosome 4** into three immortal cell

lines (HeLa, J82, T98G) assigned to complementation group B resulted in loss of proliferation and reversal of the immortal phenotype. No effect on the proliferation potential of cell lines representative of the other complementation groups was observed. This result suggests that a gene(s) involved in cellular senescence and normal growth regulation resides on **chromosome 4.**

4/7/145 (Item 11 from file: 73)
DIALOG(R)File 73:EMBASE

10532266 EMBASE No: 1999417025
Assessing linkage of monoamine oxidase B in a genome-wide scan using a univariate variance components approach
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Genetic Epidemiology (GENET. EPIDEMIOL.) (United States) 1999, 17/SUPPL. 1 (S49-S54)
CODEN: GENYE ISSN: 0741-0395
DOCUMENT TYPE: Journal; Conference Paper
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 16

We report results when one alcoholism related quantitative trait, monoamine oxidase B (MAOB), is analyzed by the variance components approach for linkage [Amos, 1994; Amos et al., 1996] using the Collaborative Study on the Genetics of Alcoholism data set provided for the Genetic Analysis Workshop 11. We used two different covariate models, one with **age** at interview, sex, ethnicity, and smoking status and the other with **age** at interview, sex, and ethnicity. The univariate analysis showed 24 markers on four different chromosomes (1, 4, 9, and 12) to have evidence for linkage with the quantitative trait (single-point and multipoint linkage). However, when outliers for MAOB were removed, the significant evidence for linkage disappeared.

4/7/214 (Item 10 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10529080 20143369 PMID: 10677304
Localization of the gene for a novel autosomal recessive neurodegenerative Huntington-like disorder to 4p15.3.
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American journal of human genetics (UNITED STATES) Feb 2000, 66 (2) p445-52, ISSN 0002-9297 Journal Code: 3IM
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
A consanguineous family affected by an autosomal recessive, progressive neurodegenerative Huntington-like disorder, was tested to rule out juvenile-onset Huntington disease (JHD). The disease manifests at approximately 3-4 years and is characterized by both pyramidal and extrapyramidal abnormalities, including chorea, dystonia, ataxia, gait instability, spasticity, seizures, mutism, and intellectual impairment. Brain magnetic resonance imaging (MRI) findings include progressive frontal cortical atrophy and bilateral caudate atrophy. Huntington CAG trinucleotide-repeat analyses ruled out JHD, since all affected individuals had repeat numbers within the normal range. The presence of only four